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Physiological Evidence That the Critical Torque Is a Phase Transition, Not a Threshold

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ABSTRACT

PETHICK, J., S. L. WINTER, and M. BURNLEY. Physiological Evidence That the Critical Torque Is a Phase Transition, Not a Threshold. *Med. Sci. Sports Exerc.*, Vol. 52, No. 11, pp. 2390–2401, 2020. **Introduction:** Distinct physiological responses to exercise occur in the heavy- and severe-intensity domains, which are separated by the critical power or critical torque (CT). However, how the transition between these intensity domains actually occurs is not known. We tested the hypothesis that CT is a sudden threshold, with no gradual transition from heavy- to severe-intensity behavior within the confidence limits associated with the CT. **Methods:** Twelve healthy participants performed four exhaustive severe-intensity trials for the determination of CT, and four 30-min trials in close proximity to CT (one or two SE above or below each participant's CT estimate; CT − 2, CT − 1, CT + 1, CT + 2). Muscle O_2 uptake, rectified electromyogram, and torque variability and complexity were monitored throughout each trial, and maximal voluntary contractions (MVC) with femoral nerve stimulation were performed before and after each trial to determine central and peripheral fatigue responses. **Results:** The rates of change in fatigue-related variables, muscle O_2 uptake, electromyogram amplitude, and torque complexity were significantly faster in the severe trials compared with CT − 2. For example, the fall in MVC torque was -1.5 ± 0.8 N·m·min^{-1} in CT − 2 versus -7.9 ± 2.5 N·m·min^{-1} in the lowest severe-intensity trial ($P < 0.05$). Individual analyses showed a low frequency of severe responses even in the circa-CT trials ostensibly above the CT, but also the rare appearance of severe-intensity responses in all circa-CT trials. **Conclusions:** These data demonstrate that the transition between heavy- and severe-intensity exercise occurs gradually rather than suddenly. **Key Words:** MUSCLE, METABOLIC RATE, NONLINEAR DYNAMICS, FATIGUE

APPLIED SCIENCES

Critical torque (CT), the asymptote of the hyperbolic relationship between isometric joint torque and the time to task failure, is an important functional threshold (1,2). Above the CT (or, analogously, the critical power [CP]), it is not possible to achieve a metabolic steady state, in contrast to exercise performed below it (for reviews, see Jones et al. [3] and Poole et al. [4]). This metabolic non–steady state is also associated with the development of peripheral fatigue at a rate proportional to the proximity of the torque or power requirements to the CT or CP (2), with peripheral fatigue developing ~4–5 times faster above CT compared with that below (2,5). At task failure during contractions above CT, the degree of peripheral fatigue is similar irrespective of the duration of the task, suggesting that both the mechanisms of fatigue and the factors resulting in task failure are common at these contractile intensities (2,5).

Many physiological variables evidence a threshold change in behavior once CT/CP is exceeded, including muscle and pulmonary $\dot{V}O_2$ (6,7), muscle phosphorylcreatine, inorganic phosphate concentration and pH (8), blood and muscle (lactate) (9), and muscle fatigue–related variables (2,5,10). Our most recent work has suggested that CT also represents a critical threshold for the fatigue-induced loss of knee extensor torque output complexity (i.e., the structure of fluctuations in the torque time series) (5). In both health and disease, physiological time series complexity has been suggested to be a key marker of system adaptability (11). In the context of the neuromuscular system, a loss of complexity in torque or force time series with aging, disease, or fatigue has been interpreted as a loss of motor control (5,12). That it is only above the CT that the complexity of muscle torque is lost as fatigue develops suggests that torque complexity also represents a sensitive marker of the proximity to the CT (5). Thus, from this perspective, the absence of steady state behavior in muscle metabolism and muscle $\dot{V}O_2$ ($m\dot{V}O_2$) above the CT/power is mirrored by a measurable loss of neuromuscular control. Collectively, therefore, measures of muscle activity, metabolism, fatigue, and time series complexity can, individually and/or collectively, provide sensitive physiological signatures of the proximity of the exercise task to the CT/power.

The threshold nature of the CT is well established, but the nature of the threshold itself is not. Although CT or CP can

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be expressed to the nearest newton-meter or watt, the confidence limits associated with the point estimate are, at best, 5%–10% of the value of CT/CP (2,10,13). As a result, the intensity at which heavy exercise responses should cease to be observed is unclear, and investigators usually, and justifiably, prescribe exercise tasks outside these confidence limits to avoid such uncertainty. Studies that have been performed at this critical threshold have returned equivocal results, with steady-state (6) and non–steady-state responses (14–16) being observed. However, by definition, performing exercise exactly at the CP or CT is itself subject to the uncertainty in the parameter estimate: 50% of responses are likely to be above CP, and 50% below (17). We state "likely" here because this is, in fact, supposition: the occurrence of heavy- or severe-intensity behavior in close proximity to CT/CP has not yet been systematically studied.

Distinguishing between behaviors that are consistent with heavy-intensity exercise versus those that are demonstrably severe is, theoretically, straightforward because tasks performed below the CT should be fatigueless (1). However, in two separate studies, we have demonstrated that fatigue does develop below CT, albeit at a very slow rate (2,5). Moreover, exercise in the heavy domain is associated with attainment of a delayed steady state, such that variables will demonstrate a positive rate of change over time when measured after the initial transient response (17). Nevertheless, the 4- to 5-fold faster rates of change observed in metabolic and fatigue-related variables above versus below the CT, coupled with consistent responses observed below the CT (2,5,8), provide a clear framework within which to study exercise responses in relation to the CT. Figure 1 illustrates purpose of the present investigation, namely, to characterize the physiological responses to tasks performed in close proximity to the CT, for which there are currently no data from our intermittent isometric contraction model.

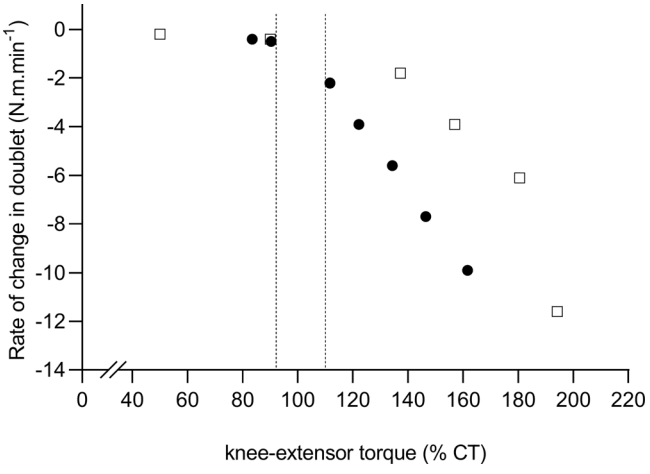

FIGURE 1—The rate of change in potentiated doublet torque as a function of the CT in two previous studies. The *black circles* are drawn from Burnley et al. (2), and the *white squares* are drawn from Pethick et al. (5). The *dashed lines* represent the limits of present observations for sub- and supra-CT exercise. Note the very slow decline in the potentiated doublet (and thus the slow development of peripheral fatigue) below the CT, but the increasingly rapid decline in doublet torque above the CT. The primary aim of the present study was to fill the void between the illustrated two dashed lines.

There are, we contend, two possible patterns of behavior that could occur in close proximity to the CT: 1) if the CT is a precise threshold, and the confidence limits associated with the parameter estimate reflect fitting error in the severe-intensity predicting trials, then consistent severe-intensity behavior should be observed in close proximity to, but nonetheless above, the CT; 2) if CT is a precise threshold that varies day-to-day by an amount represented by the confidence limits, or if the CT is itself "smeared" across a band of torque or power output and cannot be said to represent a single point value, then a small range of torque requirements exist in which the physiological response is fundamentally unpredictable and highly sensitive to the initial state of the system. In this case, the CT would behave as a continuous phase transition rather than a threshold (for a review of these general concepts, see Gisiger [18]). In other words, in close proximity to CT, severe-intensity behavior would be observed where heavy-intensity responses should be seen (<CT), and heavy-intensity responses where severe-intensity responses would be expected (>CT).

The purpose of the present study was therefore to determine whether the CT is a sudden threshold or a gradual transition from heavy to severe-intensity exercise. The specific experimental hypothesis tested was that the CT represents a sudden threshold, with no gradual transition from heavy- to severe-intensity behavior within the confidence limits associated with the CT.

METHODS

Participants

Twelve healthy participants (7 male, 5 female; mean ± SD: age, 24.7 ± 4.8 yr; height, 1.74 ± 0.09 m; body mass, 69.1 ± 11.7 kg) provided written informed consent to participate in the study, which was approved by the ethics committee of the University of Kent (Prop 10_2017_18), conformed to the standards set by the Declaration of Helsinki, except for registration in a database. Participants were instructed to arrive at the laboratory in a rested state (having performed no strenuous exercise in the preceding 24 h) and to have consumed neither any food nor caffeinated beverages in the 3 h before arrival. Participants attended the laboratory at the same time of day (±2 h) during each visit.

Experimental Design

Participants were required to visit the laboratory on nine occasions over a 5- to 9-wk period, with a minimum of 48 h between each visit. During their first visit, participants were familiarized with all testing equipment and procedures, and the settings for the dynamometer and stimulator were recorded. During visits 2–5, participants performed a series of intermittent isometric contractions to task failure (Severe Trials). From these four tests, the CT was calculated, and participants subsequently performed four further tests, during visits 6–9, just below and just above the calculated CT

(Circa-CT Trials) for 30 min or until task failure, whichever occurred sooner. In each trial, torque output was sampled continuously to allow the quantification of complexity, muscle activity was measured using the vastus lateralis electromyogram (EMG), muscle oxygen consumption ($m\dot{V}O_2$) was measured using near-infrared spectroscopy (NIRS), and maximal voluntary contractions (MVCs) with supramaximal femoral nerve stimulation were used to quantify global, central, and peripheral fatigue.

Dynamometry

During all visits, participants were seated in the chair of a Cybex isokinetic dynamometer (HUMAC Norm; CSMi, Lawrence, MA), initialized and calibrated according to the manufacturer's instructions. Their right leg was attached to the lever arm of the dynamometer, with the seating position adjusted to ensure that the lateral epicondyle of the femur was in line with the axis of rotation of the lever arm. Participants sat with relative hip and knee angles of 85° and 90°, respectively, with full extension being 0°. The lower leg was securely attached to the lever arm above the malleoli with a padded Velcro strap, whereas straps secured firmly across both shoulders and the waist prevented any extraneous movement and the use of the hip extensors during the isometric contractions. The seating position was recorded during the first visit and replicated during each subsequent visit.

Femoral Nerve Stimulation

Electrical stimulation of the right femoral nerve was used to assess neuromuscular fatigue processes, in the same way as described by Pethick (12). The anode, a carbon rubber electrode with adhesive gel (100 × 50 mm; Phoenix Healthcare Products Ltd., Nottingham, United Kingdom), was placed lateral to the ischial tuberosity, on the posterior aspect of the leg. The position of the cathode was established using a motor point pen (Compex; DJO Global, Guildford, United Kingdom) and determined based on the location in the femoral triangle, giving the largest twitch and greatest peak-to-peak amplitude of the compound muscle action potential (M-wave) after single stimulation at 100 mA, using a constant-current variable voltage stimulator (Digitimer DS7AH, Welwyn Garden City, United Kingdom). After the establishment of the precise cathode location, an Ag/AgCl electrode (32 × 32 mm; Nessler Medizintechnik, Innsbruck, Austria) coated in conductive gel was placed over the femoral nerve.

The appropriate stimulator current was then established by incrementally increasing the current (in steps of 20 mA) until knee extensor torque and the M-wave response to single twitches had plateaued. This was verified with stimulation delivered during a contraction at 50% MVC to ensure a maximal M-wave during an isometric contraction was also evident. Once this was obtained, the stimulator current was then increased to 130% of the current producing a maximal M-wave (mean ± SD, 338 ± 88 mA). In all subsequent trials, doublet stimulation (two 200-μs pulses with 10-ms interpulse interval) was used.

Surface EMG

The EMG of the vastus lateralis was sampled using Ag/AgCl electrodes (32 × 32 mm; Nessler Medizintechnik). Before attachment of the electrodes, the skin of the participants was shaved, abraded, and then cleaned with an alcohol swab over the belly of the muscle, in order to reduce impedance. The electrodes were placed on the prepared skin over the belly of the muscle in a direction parallel to the alignment of the muscle fibers. A reference electrode was placed on the prepared skin medial to the tibial tuberosity. Care was taken to ensure that the electrode locations were similar between sessions by visual inspection and palpation of the muscle belly during a light contraction. The raw EMG signals were sampled at 1 kHz, amplified (gain 1000) and band-pass filtered (10–500 Hz; Biopac MP 150; Biopac Systems Inc., Goleta, CA).

Muscle Oxygen Consumption

The $m\dot{V}O_2$ from the vastus lateralis was obtained using a continuous-wave NIRS device (Oxymon Mk III; Artinis Medical Systems, Elst, the Netherlands), calibrated according to the manufacturer's instructions before each trial. The NIRS device generated light at three wavelengths (905, 850, and 770 nm) corresponding to the absorption wavelengths of oxyhemoglobin (O_2Hb) and deoxyhemoglobin (HHb). An area at the level of the largest circumference of the vastus lateralis was shaved, abraded, and cleaned with an alcohol swab. The NIRS optode was then placed at this location and secured with Velcro straps and biadhesive tape, such that the optode did not move during contraction. A blood pressure cuff (Hokanson E20 cuff inflator; D.E. Hokanson Inc., Bellevue, WA) was placed proximal to the NIRS optode and was used to maintain blood volume under the optode during measurement. NIRS data were collected at 10 Hz. Adipose tissue thickness at the site of measurement was assessed, as per the recommendations of Ferrari et al. (19), using skinfold calipers. However, as demonstrated by Ryan et al. (20), an ischemic calibration eliminates any effect of adipose tissue thickness and scales the NIRS signals according to the maximal physiological range.

Protocol

All visits followed a pattern of data acquisition similar to that previously reported (21). Each visit began with the instrumentation of the participants and the establishment of the correct dynamometer seating position and supramaximal stimulation response. Participants then performed a series of brief (3-s) MVCs to establish their maximal torque. These contractions were separated by a minimum of 60-s rest and continued until the peak torques in three consecutive contractions were within 5% of each other. Participants were given a countdown, followed by very strong verbal encouragement to maximize torque. The first MVC was used to establish the fresh maximal EMG signal, against which the subsequent EMG signals were normalized (Data Analysis). The second and third MVCs were performed with femoral nerve stimulation delivered during the contraction

and at rest after the contraction. The stimulation during the contraction was delivered during a plateau in maximal torque, in order to test the maximality of the contraction and provide the resting voluntary activation, whereas the stimulation at rest was delivered 2 s after the contraction, in order to establish the fresh potentiated doublet torque. All subsequent contractions with femoral nerve stimulation were conducted in this manner.

After the establishment of maximal torque, the resting $m\dot{V}O_2$ of the vastus lateralis was assessed based on the decrease in muscle oxygenation, which accompanies an arterial occlusion (20,22). For this, the blood pressure cuff was inflated to 300 mm Hg using a Hokanson AG101 (D.E. Hokanson Inc.). Four resting measurements were made using 10 s of arterial occlusion, each separated by 60-s rest. The resting $m\dot{V}O_2$ was calculated using linear regression with the first 8 s of each occlusion (Data Analysis). Participants then rested for 10 min before performing the experimental trial (see below).

Severe Trials

During visit 2 (the first of the severe trials), the highest instantaneous pretest measure of voluntary torque was recorded as the peak MVC torque, and the target torques for the submaximal contractions in visits 2–5 were calculated from this value. The submaximal contractions were performed, as in our previous studies (5,12) using a duty cycle of 0.6, with contractions held for 6 s, followed by 4-s rest. The target for the submaximal contractions in visit 2 was set at 50% of the peak torque measured in the pretest MVCs. Participants were instructed to match their instantaneous torque with a target bar superimposed on the display in front of them and were required to continue matching this torque for as much of the 6-s contraction as possible. The test was conducted until task failure, the point at which the participant failed to reach the target torque on three consecutive contractions, despite strong verbal encouragement. Participants were not informed of the elapsed time during the test, but were informed of each "missed" contraction. After the third missed contraction, participants were instructed to produce an MVC, which was accompanied by peripheral nerve stimulation.

At the end of each minute (i.e., after every fifth contraction), $m\dot{V}O_2$ was assessed, as described by Pethick et al. (21). The blood pressure cuff was inflated to 300 mm Hg for 5 s, with $m\dot{V}O_2$ calculated using linear regression over the course of this occlusion. This measure of $m\dot{V}O_2$ was performed instead of a targeted contraction. $m\dot{V}O_2$ was also assessed immediately before the MVC performed at task end/failure. Five minutes after task end/failure, an ischemia/hyperemia calibration was performed to normalize the NIRS signals. The blood pressure cuff was inflated to 300 mm Hg for 3–5 min (or until the NIRS signals plateaued). This deoxygenated the tissue under the optode (defined as 0% oxygenation), whereas the peak hyperemic response upon release of the cuff was assigned a value of 100% oxygenation.

The duration of the initial severe trial at 50% MVC was used to determine the percentage of MVC used in the subsequent three trials, which were performed in an identical manner. The objective of these trials was to yield times to task failure of between 2 and 15 min, which have been recommended for the assessment of CT (23). The subsequent severe trials were performed in a randomized order. Visits 2–5 were used to determine the CT. The individual trials were identified as *severe 1* (S1) to *severe 4* (S4), with S1 and S4 being the lowest and highest torques, respectively.

Circa-CT Trials

The final four visits were performed at target torques based on the SE of the calculated CT. The target torques were two SE below the CT (CT − 2), one SE below the CT (CT − 1), one SE above the CT (CT + 1), and two SE above the CT (CT + 2). Thus, by design, each of these trials was within the 95% confidence limits of the CT parameter estimate, but below or above the point estimate. These trials were conducted in the same manner as the severe trials, requiring participants to perform intermittent contractions (6-s on, 4-s off) at a target torque. In these trials, the contractions were performed for 30 min or until task failure, whichever occurred sooner. These four *circa-CT* trials were performed in a randomized order. No trials were performed exclusively below CT because of the already heavy time commitment on the part of the participants (nine laboratory visits of up to 2 h each), so as to maximize the granularity of the circa-CT analysis. As a result, the responses to the circa-CT trials were compared with our previously published studies (Fig. 1) to establish whether those response profiles approximated heavy exercise.

Data Acquisition and Participant Interface

Data acquisition was performed in a similar manner as previously described (21). Briefly, the isokinetic dynamometer, stimulator and EMG were connected via BNC cables to a Biopac MP150 (Biopac Systems Inc.) and a CED Micro 1401–3 (Cambridge Electronic Design, Cambridge, United Kingdom) interfaced with a personal computer. These data were sampled at 1 kHz and collected in Spike2 (Version 7; Cambridge Electronic Design). The NIRS data were sampled at 10 Hz and collected in OxySoft (Artinis Medical Systems).

A chart containing the instantaneous torque was projected onto a screen placed ~1 m in front of the participant. A scale consisting of a thin line (1 mm thick) was superimposed on the torque chart and acted as a target, so that participants were able to match their instantaneous torque output to the target torque during each visit.

Data Analysis

All data were analyzed using a code written in MATLAB R2017a (The MathWorks, Natick, MA). The data analysis focused on five areas: 1) basic measures of torque and EMG, 2) calculation of CT, 3) measures of central and peripheral fatigue, 4) the variability and complexity of torque output, and 5) measures of $m\dot{V}O_2$.

Torque and EMG. The mean and peak torque for each contraction in each trial were determined. The mean torque was calculated based on the steadiest 5 s of each contraction, with MATLAB code identifying the 5 s of each contraction with the lowest SD. The point of task failure was determined as described previously (12). The mean torque produced during the first five contractions was calculated, with task failure deemed to occur when the mean torque recorded during three consecutive contractions was more than 5 N·m below the mean torque of the first five contractions, with the first of these contractions being considered the point of task failure.

The EMG output from the vastus lateralis for each contraction was full-wave rectified during each 5-s window. The average rectified (arEMG) was then calculated and normalized by expressing the arEMG as a fraction of the arEMG obtained during an MVC from the fresh muscle performed at the beginning of the trial.

CT. CT was determined as in Pethick et al. (5). The total torque impulse produced until task failure and the total contraction time during each individual trial were calculated. The torque impulse was then plotted against the contraction time, and the parameters of the torque–duration relationship were estimated using linear regression of the torque impulse versus contraction time (2):

$$\text{torque impulse} = W' + \text{CT} \times t \quad [1]$$

where W' represents the curvature constant parameter and t is the time to task failure.

Central and peripheral fatigue. Measures of central and peripheral fatigue were calculated based on the stimuli delivered during and after the MVCs performed pretest and at task end/failure. Peripheral fatigue was evidenced by a fall in the potentiated doublet torque. Central fatigue was evidenced by a decline in voluntary activation, quantified using the twitch interpolation technique (24,25):

$$\text{voluntary activation (\%)} = (1 - \text{superimposed doublet/resting doublet}) \times 100 \quad [2]$$

where the superimposed doublet was that measured during the contraction of interest and the potentiated doublet was measured at rest 2 s after that contraction.

Variability and complexity. All measures of variability and complexity were calculated using the steadiest 5 s of each contraction, identified by MATLAB as the 5 s containing the lowest SD. The amount of variability in the torque output of each contraction was measured using the SD and coefficient of variation (CV). The SD provides a measure of the absolute amount of variability in a time series, whereas the CV provides a measure of the amount of variability in a time series normalized to the mean of the time series.

The temporal structure, or complexity, of torque output was examined using multiple time domain analyses. The regularity of torque output was determined using approximate entropy (ApEn) (26), and the temporal fractal scaling of torque was estimated using the detrended fluctuation analysis (27) α scaling exponent (DFA α). The calculations of ApEn and DFA are detailed in the study by Pethick et al. (21). In brief, ApEn was calculated with the template length, m, set at 2 and the tolerance, r, set at 10% of the SD of torque output, and DFA was calculated across time scales (57 boxes ranging from 1250 to 4 data points). If crossover in the DFA analysis was identified in the log–log plot of fluctuation size versus box size (as shown by an $r < 0.95$), an iterative piecewise least squares linear regression was used to fit two lines to the plot for these trials, and two α exponents were quantified. The second of these (α2, representing longer, physiologic timescales) was used in the DFA α exponent analysis (21). Ten cases of crossover occurred during the circa-CT trials (out of 48 trials) and none during the severe-intensity predicting trials.

Muscle oxygen consumption. $m\dot{V}O_2$ was determined as described by Ryan et al. (20,22). $m\dot{V}O_2$ was calculated as the slope of the change in O_2Hb and HHb during arterial occlusion using simple linear regression. The resting $m\dot{V}O_2$ measurement was based on the first 8 s (80 data points) of a 10-s arterial occlusion, whereas the exercising $m\dot{V}O_2$ measurements were based on a 5-s arterial occlusion (50 data points).

The NIRS data were corrected for blood volume changes (20,22) using custom-written MATLAB code. A blood volume correction factor (β) was calculated for each data point during the arterial occlusions:

$$\beta(t) = \frac{|O_2Hb(t)|}{(|O_2Hb(t)| + |HHb(t)|)} \quad [3]$$

where β is the blood volume correction factor, t is time, O_2Hb is the oxygenated hemoglobin/myoglobin signal, and HHb is the deoxygenated hemoglobin/myoglobin signal. Each data point was corrected using its corresponding β according to Eqs. 4 and 5, as follows:

$$O_2Hb_c(t) = O_2Hb(t) - [tHb(t) \times (1 - \beta)] \quad [4]$$

$$HHb_c(t) = HHb(t) - [tHb(t) \times \beta] \quad [5]$$

where O_2Hb_c and HHb_c are the corrected oxygenated and deoxygenated hemoglobin/myoglobin signals, respectively; tHb is the blood volume signal from the NIRS device; β is the blood volume correction factor; and t is time. The raw O_2Hb signal in equation 4 is corrected by subtracting the proportion of the blood volume change attributed to O_2Hb, whereas in equation 5, the raw HHb signal is corrected by subtracting the proportion of blood volume change attributed to HHb.

Rates of Change

To identify heavy- or severe-intensity behavior in $m\dot{V}O_2$, arEMG and fatigue and complexity-related variables, we first calculated the rate of change in CT − 2 from the second minute of exercise until task failure or task completion (30 min)—in order to avoid contamination of the primary kinetics of $m\dot{V}O_2$—and compared this with the equivalent rates of change in all other circa-CT trials. Specifically, the rate of change in CT − 2 for each individual participant was subtracted from their rates of change in each other condition, assuming that

TABLE 1. Voluntary torque, potentiated doublet torque, and voluntary activation during contractions within the confidence limits of (CT – 2, CT – 1, CT + 1, and CT + 2) and above (S1–S3) the CT.

Parameter	CT – 2	CT – 1	CT + 1	CT + 2	S1	S2	S3
Target torque, N·m	57.2 ± 15.1	59.3 ± 15.1	63.5 ± 15.3	65.6 ± 15.5	85.5 ± 21.6	97.4 ± 25.0	112.2 ± 26.5
Target torque, % MVC	24.8 ± 3.1	25.7 ± 3.0	27.6 ± 3.0	28.6 ± 3.0	37.1 ± 3.5	42.1 ± 3.3	48.8 ± 3.1
Time to task end/failure, min	30.0 ± 0.0	30.0 ± 0.0	30.0 ± 0.0	29.5 ± 1.7	16.8 ± 2.8	9.7 ± 1.2	6.2 ± 0.9
Global fatigue							
Preexercise MVC, N·m	245.8 ± 65.8	245.7 ± 71.1	247.2 ± 63.5	243.3 ± 63.5	236.3 ± 71.3	233.6 ± 62.1	233.9 ± 66.6
Peak MVC task end, N·m	197.8 ± 47.4	189.4 ± 58.6	172.2 ± 55.4	156.9 ± 56.7[a]	106.5 ± 44.0[a]	112.7 ± 45.2[a]	127.7 ± 41.5[a]
Mean MVC task end, N·m	167.6 ± 42.9[b,c]	162.1 ± 54.9[c]	147.1 ± 49.2[b,c]	133.3 ± 50.4[b,c]	86.0 ± 35.0[b]	98.8 ± 41.1[b]	108.7 ± 38.9[b]
ΔMVC/Δ*t*, N·m·min^{-1}	–1.5 ± 0.8	–1.7 ± 0.7	–2.4 ± 0.9[a]	–2.9 ± 1.7	–7.9 ± 2.5[a]	–12.5 ± 3.4[a]	–17.1 ± 6.6[a]
Peripheral fatigue							
Preexercise doublet, N·m	93.5 ± 31.1	99.1 ± 33.0	96.7 ± 31.9	93.1 ± 27.0	94.4 ± 32.3	94.0 ± 28.4	91.2 ± 33.4
Doublet task end, N·m	87.7 ± 29.2[b]	92.9 ± 32.6[b]	83.9 ± 28.3[b]	79.8 ± 25.3[a,b]	66.4 ± 23.5[a,b]	67.6 ± 27.2[a,b]	65.6 ± 27.2[a,b]
% Change task end	6.0 ± 4.8	6.4 ± 4.9	13.3 ± 3.7	14.2 ± 6.9	29.3 ± 9.0	29.4 ± 20.6	27.5 ± 7.5
Δdoublet/Δ*t*, N·m·min^{-1}	–0.2 ± 0.2	–0.2 ± 0.2	–0.4 ± 0.2[a]	–0.4 ± 0.3[a]	–1.7 ± 0.8[a]	–2.7 ± 1.1[a]	–4.3 ± 2.6[a]
Central fatigue							
Preexercise VA, %	95.5 ± 3.7	94.7 ± 2.6	94.5 ± 3.4	94.7 ± 3.4	94.9 ± 2.7	93.3 ± 2.6	93.8 ± 3.0
VA task end, %	92.2 ± 3.6[b]	90.9 ± 5.1	84.9 ± 7.2[b]	85.6 ± 9.0[b]	77.1 ± 10.8[a,b]	80.5 ± 9.2[a,b]	80.3 ± 8.3[a,b]
% Change task end	3.5 ± 3.0	3.9 ± 4.6	10.1 ± 7.1	9.6 ± 9.0	18.8 ± 11.2	13.6 ± 10.6	14.6 ± 7.3
ΔVA/Δ*t*, %/min	–0.1 ± 0.1	–0.1 ± 0.1	–0.3 ± 0.2	–0.3 ± 0.3	–1.1 ± 0.7[a]	1.3 ± 1.0[a]	–2.3 ± 1.2[a]

Values are means ± SD.
Symbols indicate a statistically significant difference compared with CT – 2, value at task beginning, and target torque.
[a]CT – 2 ($P < 0.05$).
[b]Value at task beginning.
[c]Target torque.
Δ, change; *t*, time; VA, voluntary activation.

CT − 2 most likely represented heavy-intensity exercise. Severe-intensity exercise was then deemed to have occurred if the resulting "normalized" rate of change was 2 SD greater than that individual's CT − 2 value, where the SD was the SD of the rate of change in CT − 2 (as reported in Tables 1 and 2). Thus, if a participant's responses to CT − 1, CT + 1, and CT + 2 exceeded the 2-SD criterion, they were considered severe intensity, but if below it, they were considered heavy. We accept that this value is arbitrary, but in the absence of any agreed quantitative definition of heavy-intensity behavior, we suggest that this "2 σ" approach provides an objective and justifiable metric. Using criterion values greater than 2 SD is likely to result in misclassification (i.e., misclassifying severe-intensity exercise as heavy).

Statistics

All data are presented as means ± SD unless otherwise stated. The S4 trial was used only for the purposes of determining CT and was excluded from all further analysis. This was due to its relatively short duration making the calculation of rates of change impossible. Two-way ANOVA with repeated

TABLE 2. EMG amplitude, muscle oxygen uptake, variability, complexity, and fractal scaling responses during contractions within the confidence limits of (CT – 2, CT – 1, CT + 1, and CT + 2) and above (S1–S3) the CT.

Parameter	CT – 2	CT – 1	CT + 1	CT + 2	S1	S2	S3
arEMG start, % MVC	27.3 ± 7.2	27.3 ± 5.6	32.0 ± 9.0	32.5 ± 6.6	42.4 ± 7.6	47.4 ± 8.7	61.6 ± 12.7
arEMG task end, % MVC	31.5 ± 8.1	34.8 ± 8.7	41.9 ± 12.0[a,b]	48.4 ± 18.2[a,b]	68.7 ± 17.2[a,b]	73.4 ± 13.9[a,b]	80.8 ± 10.8[a,b]
ΔarEMG/Δ*t*, % MVC/min	0.2 ± 0.2	0.3 ± 0.3	0.4 ± 0.3[b]	0.6 ± 0.6	1.8 ± 1.1[b]	3.5 ± 1.4[b]	4.8 ± 3.2[b]
$m\dot{V}O_2$ start, %·s^{-1}	1.65 ± 0.70	1.86 ± 0.80	2.24 ± 0.98	2.45 ± 0.87	2.43 ± 0.99	2.64 ± 1.12	2.60 ± 0.96
$m\dot{V}O_2$ at task end, %·s^{-1}	1.98 ± 1.00[a]	2.07 ± 0.92[a]	2.67 ± 1.12[a,b]	3.10 ± 0.85[a,b]	3.27 ± 1.21[a,b]	3.57 ± 1.21[a,b]	3.33 ± 0.94[a,b]
$\Delta m\dot{V}O_2/\Delta t$, %·s^{-1}	0.01 ± 0.01	0.01 ± 0.01	0.02 ± 0.01	0.03 ± 0.01	0.06 ± 0.02[b]	0.12 ± 0.08[b]	0.19 ± 0.08[b]
SD							
SD at task beginning, N·m	1.33 ± 0.45	1.30 ± 0.44	1.37 ± 0.32	1.33 ± 0.32	1.87 ± 0.54	2.24 ± 0.51	2.95 ± 1.07
SD at task failure, N·m	1.64 ± 0.62[a]	1.55 ± 0.37[a]	1.87 ± 0.49[a]	1.90 ± 0.80[a]	4.71 ± 2.34[a,b]	7.71 ± 1.21[a,b]	7.85 ± 2.81[a,b]
ΔSD/Δ*t*, N·m·min^{-1}	0.01 ± 0.01	0.01 ± 0.01	0.02 ± 0.01	0.02 ± 0.03	0.19 ± 0.13[b]	0.43 ± 0.35[b]	1.20 ± 0.59[b]
CV							
CV at task beginning, %	2.30 ± 0.68	2.27 ± 0.66	2.13 ± 0.41	2.03 ± 0.47	2.30 ± 0.65	2.43 ± 0.64	2.66 ± 0.61
CV at task failure, %	2.77 ± 1.04[a]	2.65 ± 0.55[a]	2.98 ± 0.66[a]	2.84 ± 1.18[a]	6.01 ± 2.16[a,b]	7.71 ± 1.21[a,b]	8.29 ± 4.24[a,b]
ΔCV/Δ*t*, %/min	0.02 ± 0.02	0.01 ± 0.02	0.03 ± 0.02	0.03 ± 0.01	0.21 ± 0.14[b]	0.48 ± 0.30[b]	1.38 ± 0.94[b]
ApEn							
ApEn at task beginning	0.56 ± 0.17	0.60 ± 0.24	0.55 ± 0.19	0.56 ± 0.14	0.46 ± 0.15	0.41 ± 0.14	0.32 ± 0.07
ApEn at task failure	0.51 ± 0.18[a]	0.54 ± 0.19[a]	0.45 ± 0.10[a]	0.44 ± 0.17[a]	0.16 ± 0.09[a,b]	0.15 ± 0.07[a,b]	0.07 ± 0.03[a,b]
ΔApEn/Δ*t*	–0.002 ± 0.002	–0.002 ± 0.003	–0.001 ± 0.01	–0.005 ± 0.004	–0.02 ± 0.01[b]	–0.03 ± 0.01[b]	–0.6 ± 0.3[b]
DFA α							
DFA α at task beginning	1.40 ± 0.06	1.38 ± 0.10	1.39 ± 0.06	1.37 ± 0.07	1.40 ± 0.06	1.40 ± 0.05	1.45 ± 0.05
DFA α at task failure	1.35 ± 0.10[a]	1.22 ± 0.25[a]	1.22 ± 0.25[a]	1.33 ± 0.17	1.56 ± 0.04[a,b]	1.57 ± 0.09[a,b]	1.65 ± 0.12[a,b]
ΔDFA α/Δ*t*	–0.002 ± 0.003	–0.006 ± 0.008	0.006 ± 0.008	0.001 ± 0.006	0.01 ± 0.01[b]	0.02 ± 0.02[b]	0.05 ± 0.04[b]

Values are means ± SD. Task start values are values from 2 min into exercise, to account for primary amplitude of $m\dot{V}O_2$ response.
Symbols indicate a statistically significant difference compared with the value at task beginning and CT – 2.
[a]Value at task beginning.
[b]CT – 2 ($P < 0.05$).
Δ, change; *t*, time.

measures was used to test for differences between conditions and time points, and for a condition–time interaction for MVC torque, arEMG, potentiated doublet torque, voluntary activation, variability, complexity, and $m\dot{V}O_2$. The variability, complexity, and $m\dot{V}O_2$ measures were analyzed using means from the second minute to account for the primary kinetics of the $\dot{V}O_2$ response (28) and final minute before task end/failure. When main effects were observed, Bonferroni-adjusted 95% paired-samples confidence intervals (CI) were used to identify specific differences. The rates of change in all variables were analyzed using Student paired-samples *t*-tests.

RESULTS

Preliminary measures and the CT. The peak instantaneous MVC torque recorded during an MVC in visit 2 was 233.4 ± 66.7 N·m. This was used to set the target torques for the tests performed above CT, which are presented in Table 1. The CT was calculated to be 61.4 ± 15.2 N·m, which was equivalent to 26.7% ± 3.0% MVC, and the W' was 4800 ± 1428 N·m·s. The 95% CI for the estimation of CT was 8.9 ± 2.8 N·m. The torque requirements for the circa-CT trials were determined as one or two SE below or above CT (i.e., within the 95% confidence limits of CT; Table 1).

Torque, muscle oxygen consumption, and EMG. For the trials above the CT, task failure occurred when participants were no longer able to achieve the target torque, despite a maximal effort. All trials above the CT resulted in significant decreases in MVC torque (F = 140.80, P < 0.001), with the mean MVC torque at task failure being not significantly different from the torque produced during the submaximal contractions (Table 1). In contrast, all participants completed 30 min of contractions (with the exception of one trial in CT + 2) in the circa-CT trials. At the end of these trials, the mean MVC torque was still significantly greater than the submaximal torque requirements (95% paired-samples CI = 89.4–131.3 N·m for CT − 2, 74.9–130.6 N·m for CT − 1, 58.9–108.3 N·m for CT + 1, 39.5–95.9 N·m for CT + 2), indicating that the contractions ended with a substantial reserve in maximal torque. MVC torque remained significantly greater at task end in CT − 2 compared with task end/failure in CT + 2 (CI = 1.5–67.1 N·m), S1 (CI = 60.9–102.4 N·m), S2 (CI = 44.0–93.6 N·m), and S3 (CI = 39.5–78.2 N·m). The rate of change in MVC torque was greater for CT + 1 and all severe-intensity trials (S1–S3) compared with CT − 2 (Table 1).

After the initial transient, all of the trials resulted in significant increases in $m\dot{V}O_2$ (F = 257.03, P < 0.001; Fig. 2). The $m\dot{V}O_2$ at task end in CT − 2 remained significantly lower compared with task end/failure in CT + 1 (CI = −1.6 to −0.04%·s^{-1}), CT + 2 (CI = −1.7 to −0.6%·s^{-1}), S1 (CI = −2.2 to −0.3%·s^{-1}), S2 (CI = −2.8 to −0.3%·s^{-1}), and S3 (CI = −2.2 to −0.5%·s^{-1}). The arEMG amplitude increased over time in all of the trials above CT, reaching ~68% to 80% of the pretest MVC value at task failure (F = 58.30,

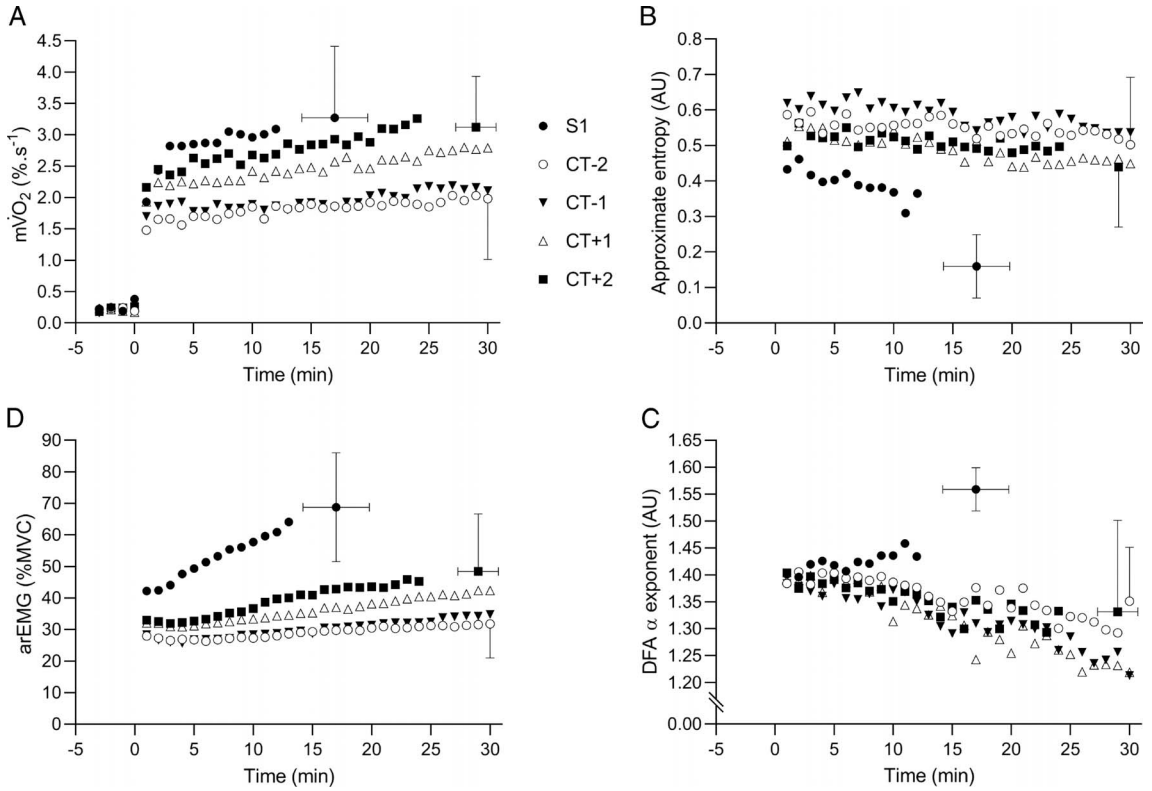

FIGURE 2—Group mean muscle oxygen consumption (A), average rectified EMG (B), ApEn (C), and DFA α exponent (D) responses to contractions close to and above CT. The four circa-CT trials are represented (CT − 2 to CT + 2), along with the lowest severe-intensity predicting trial (S1). Error bars are shown only shown at the conclusion of S1, CT − 2, and CT + 2 for clarity (n = 12).

$P < 0.001$; Fig. 2, Table 2). The circa-CT trials also resulted in increases in arEMG as the trials progressed, although these were more modest than for the trials above the CT (Table 1). The arEMG remained significantly lower at task end in CT − 2 compared with task end/failure in CT + 1 (CI = −15.5% to −5.2%), CT + 2 (CI = −25.8% to −7.9%), S1 (CI = −49.4% to −24.5%), S2 (CI = −48.6% to −35.0%), and S3 (CI = −55.3% to −43.2%). The rates of change for the circa-CT trials did not differ from CT − 2, whereas all trials above the CT had greater rates of change than CT − 2 for both $m\dot{V}O_2$ and arEMG (Table 2). Figure 3 shows the individual rates of change in $m\dot{V}O_2$ and arEMG normalized to the rates of change in the CT − 2 trial for the circa-CT trials and S1. None of the circa-CT demonstrated consistent severe-intensity behavior in either parameter (defined as a rate of change 2 SD greater than the CT − 2 mean value), although 1–5 participants exceeded this threshold value during these trials. The majority of individual responses to the S1 trial were correctly classified as severe-intensity (Figs. 3A, and 3B).

Peripheral and central fatigue. All of the trials above the CT resulted in significant reductions in potentiated doublet torque ($F = 65.60$, $P < 0.001$; Table 1), indicating the presence of peripheral fatigue. The circa-CT trials also resulted in modest, though significant, reductions in potentiated doublet torque (Table 1). The potentiated doublet torque remained significantly greater at task end in CT − 2 compared with task end/failure in CT + 2 (CI = 0.07–15.0 N·m), S1 (CI = 10.7–31.9 N·m), S2 (CI = 3.1–37.2 N·m), and S3 (CI = 5.9–33.3 N·m). Voluntary activation significantly decreased during all trials above the CT ($F = 37.02$, $P < 0.001$; Table 1),

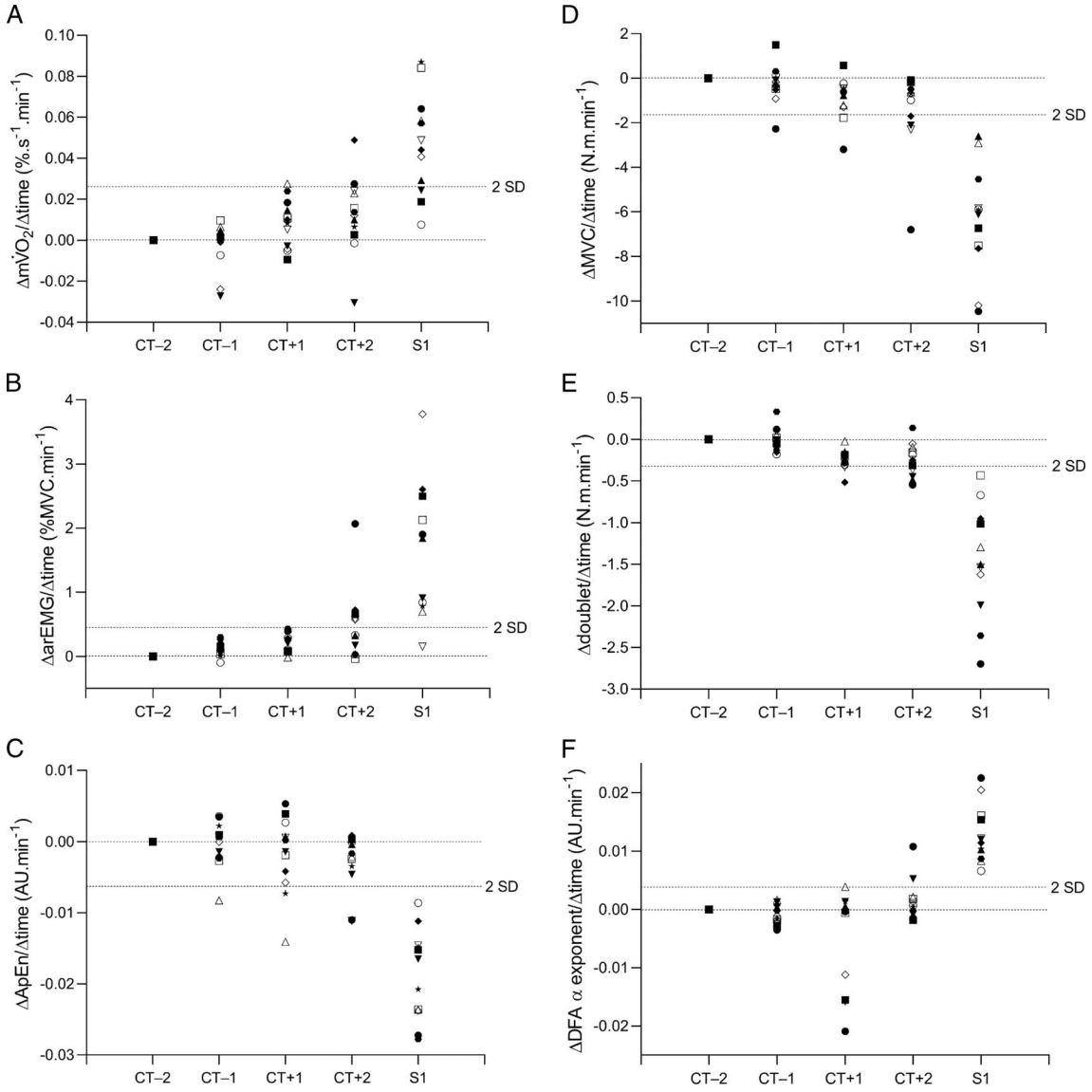

FIGURE 3—Individual rates of change in the muscle oxygen consumption (A), average rectified EMG amplitude (B), ApEn (C), the MVC torque (E), potentiated doublet torque (E), and the DFA α exponent (F) during the circa-CT trials and the S1 trial. In these plots, the rates of change across conditions are normalized to the CT − 2 condition, thereby eliminating false positives/negatives created by applying sample statistics to the raw individual responses. The two *lines* on each panel represent the normalized CT − 2 value and a change of two SD values toward severe-intensity exercise. Severe responses were estimated as being greater than 2 SD values from the normalized CT − 2 response ($n = 12$).

indicating the presence of central fatigue. The circa-CT trials also resulted in modest, though significant, decreases in voluntary activation (Table 1). Voluntary activation remained significantly greater at task end in CT − 2 compared with task failure in S1 (CI = 8.2%–28.8%), S2 (CI = 6.1%–17.1%), and S3 (CI = 7.8%–16.6%). There was a greater rate of change in potentiated doublet torque during CT + 1, CT + 2, and all other trials above the CT compared with CT − 2. The individual rates of change in the MVC and potentiated doublet torque are presented in Figure 3. As previously discussed, for $m\dot{V}O_2$ and arEMG, there were no consistent severe-intensity responses to the circa-CT trials, although severe-intensity behavior was inconsistently observed for both variables. In contrast, all participants demonstrated correctly classified severe-intensity responses to the S1 trial.

Variability and complexity. The variability and complexity data are presented in Table 2. The amount of variability, as measured by the SD ($F = 60.29$, $P < 0.001$) and CV ($F = 78.56$, $P < 0.001$), increased in all trials. The SD and CV remained significantly lower at task end in CT + 2 compared with task failure in S1 (CI = −5.2 to −0.9 N·m for SD, −5.4% to −1.1% for CV), S2 (CI = −6.0 to −1.4 N·m for SD, −5.2% to −1.3% for CV), and S3 (CI = −8.7 to −3.7 N·m for SD, −9.3% to −1.7% for CV).

Complexity decreased throughout all trials, as measured by ApEn ($F = 132.11$, $P < 0.001$) and DFA α (save CT − 2; $F = 28.98$, $P < 0.001$; Fig. 2, Table 2). ApEn decreased over the course of all severe trials but remained significantly greater at task end in CT − 2 compared with task failure in S1 (CI = 0.3–0.5), S2 (CI = 0.2–0.5), and S3 (CI = 0.3–0.6). DFA α increased over the course of all the above CT trials and remained significantly lower at task end in CT − 2 compared with task failure in S1 (CI = −0.2 to −0.1), S2 (CI = −0.3 to −0.1), and S3 (CI = −0.4 to −0.2). There was no difference in the rates of change between the circa-CT trials, but all trials above CT showed a greater rate of change than CT − 2 (Table 2). The individual rates of change are presented in Figure 3. Severe-intensity behavior was inconsistently observed during the circa-CT trials for both ApEn and DFA α, but the majority of cases appeared to be responses in the heavy domain. In contrast, all of the responses to S1 were clearly placed in the severe-intensity domain.

DISCUSSION

The principal original finding of the present study was that exercise tasks in close proximity to the CT did not show evidence of a sudden fatigue, neuromuscular, metabolic, or physiological complexity threshold at the CT, in contrast to our first hypothesis. Specifically, we did not observe consistently severe-intensity behavior in $m\dot{V}O_2$, arEMG, fatigue-related variables (MVC, potentiated doublet, and voluntary activation), or torque complexity two SE above the CT (~107% of CT). In contrast, the lowest severe-intensity task (~139% CT) showed clear evidence of severe-intensity behavior in the above variables. Nevertheless, there was some evidence of severe-intensity responses in some individuals exercising close to the CT, suggesting that the physiological responses to exercise within the confidence limits of the CT are unpredictable. The CT therefore appears to represent a band of torque values rather than a precise point estimate, akin to a form of continuous phase transition between heavy and severe intensity exercise. Thus, we provide the first evidence that the CT, although representing a metabolic and fatigue threshold in the context of separating distinct intensity domains, the transition itself is not sudden but is rather smeared across a band of torque represented by the 95% confidence limits of the CT parameter estimate.

Typical responses observed in the heavy and severe-intensity domains. There is no question that distinct physiological responses can be observed across the exercise intensity spectrum, with experimentally identifiable physiological landmarks separating at least four exercise intensity domains (7,28,29). In previous studies, we have demonstrated that exercise performed below the CT eventually results in stable responses (in arEMG), very slowly developing peripheral fatigue (2), and no change in torque output complexity (5). For tasks requiring >110% CT, distinctly different fatigue mechanisms, muscle metabolic responses, and adaptations in neuromuscular control occur (2,5,8). Specifically, severe-intensity exercise is characterized by non–steady-state metabolic and neuromuscular responses, and exercise tolerance is usually limited to less than ~30 min (e.g., Refs. [2,6,8]). All severe-intensity trials in the present experiments shared these characteristics: both $m\dot{V}O_2$ and arEMG increased systematically until task failure occurred (in less than ~20 min; Fig. 2, Table 2). The fall in MVC was such that at task failure a maximal contraction was required to produce the required torque, and the potentiated doublet torque had declined to consistently low values (Table 1). Central fatigue was also evident, with voluntary activation falling by approximately 10%, consistent with previously reported responses (2,5). The severe-intensity trials were also associated with substantial reductions in torque steadiness (torque SD or CV) and torque complexity (ApEn or DFA α exponent; Table 2, Fig. 2) as previously reported (5).

Behavior in close proximity to the CT. The central aim of the present study was to determine how these adjustments behaved in very close proximity to the CT. If the CT is a sudden threshold, with the confidence limits simply representing a fitting error in the linear regression of the torque-time integral (as a result of variance in severe-intensity performance within the predicting trials), severe-intensity behavior should have been absent in all tasks below CT and present in all tasks above CT. The present study lacked a condition that was demonstrably below the CT. This was a deliberate design choice to focus on the responses within the 95% confidence limits of the CT while keeping the demands on the participants for repeated testing to a minimum (the present experiments required nine laboratory visits lasting approximately 2 h each). Our previous work on this issue in comparable cohorts again provides a useful point of reference (2,5). The responses to the CT − 2 condition in the present study (performed at ~93% CT) were virtually identical to those responses to a trial at 90% CT

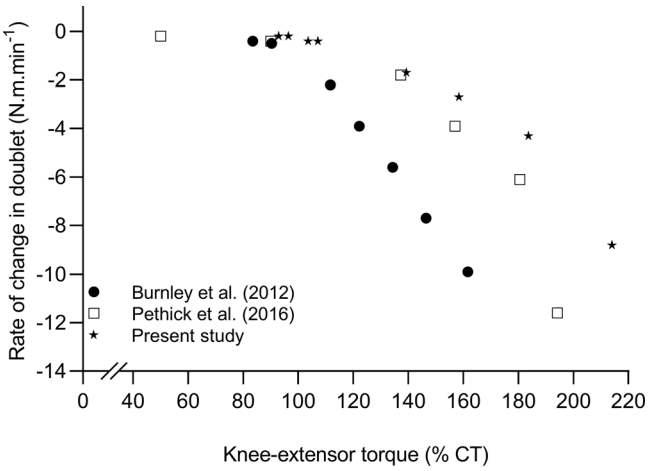

FIGURE 4—The rate of change in potentiated doublet torque as a function of the CT in the present study, plotted alongside the data from Figure 1. Data are drawn from Burnley et al. (2) (*white circles*), Pethick et al. (5) (*white squares*) and the present study (*black stars*). The intensity of each trial is plotted as a percentage of the CT in that study. Note the increase in the rate of decline in potentiated doublet torque above the CT in all studies, and the lack of a sudden decline in the potentiated doublet in close proximity to the CT (shown by the cluster of four stars at the top of the figure). This suggests that a continuous phase transition, rather than a threshold, best describes the emergence of severe-intensity behavior above the CT.

in a study by Pethick et al. (5, their Tables 1 and 2, present study's Fig. 4). For example, in our two previous studies, the rate of fall in potentiated doublet below the CT has never exceeded 0.9 $N \cdot m \cdot min^{-1}$. The largest rate of decline in this variable in CT − 2 was 0.5 $N \cdot m \cdot min^{-1}$.

In contrast to the severe-intensity responses, the CT − 2 trial was associated with only small (but statistically significant) changes in $m\dot{V}O_2$, the MVC, and voluntary activation. Each of these was significantly smaller in magnitude than those recorded in the severe-intensity trials, and at the end of the task (30 min), there was a significant reserve in torque-generating capacity (MVC). Similarly, the changes in torque steadiness and complexity, which were small but statistically significant, did not reach values observed at task failure in the severe-intensity trials. Importantly, compared with CT − 2, all severe-intensity trials showed greater fatigue-related responses at task failure (in $m\dot{V}O_2$, arEMG, MVC, potentiated doublet, VA, torque steadiness, and complexity), indicating that the severe-intensity tasks were performed in a different region of the exercise intensity spectrum than CT − 2. Thus, despite its close proximity to the CT, all pieces of the evidence, from the present and previous studies, demonstrate that CT − 2 was performed in the heavy-intensity domain.

If the CT represents a sudden threshold, then the two trials performed above the CT (CT + 1 and CT + 2) should have produced severe-intensity exercise responses. Several parameters showed statistically significant changes in the CT + 1 and/or CT + 2 trials compared with CT − 2 (Tables 1, 2). For example, MVC and potentiated doublet torque decreased to a greater degree in CT + 1 and CT + 2 compared with CT − 2 (Table 1), and $m\dot{V}O_2$ and arEMG were both significantly higher after 30 min above CT compared with CT − 2 (Table 2). In addition, the rate of increase in $m\dot{V}O_2$ was also higher in CT + 1 but not CT + 2. Finally, the rate of change in MVC torque was higher in CT + 1 than CT − 2, and the rate of change in the potentiated doublet, an index of peripheral fatigue, was higher in both CT + 1 and CT + 2. However, differences between the CT + 1 and CT + 2 trials compared with CT − 2 would be expected simply on the basis that the former trials were performed at a higher absolute torque requirement. Of greater importance is whether or not the responses observed were demonstrably in the severe-intensity domain, which would be expected if the CT is indeed a sudden physiological threshold.

In lieu of a rigorous classification for steady-state and non–steady-state behavior in the variables measured in the present study (compare the blood lactate response to exercise) (17), we attempted to further differentiate heavy- and severe-intensity exercise by considering any individual rate of change two SD values greater than that of the mean in CT − 2 to indicate severe-intensity exercise. These analyses (Fig. 3) revealed that severe-intensity behavior in $m\dot{V}O_2$, arEMG, MVC, potentiated doublet, and torque complexity could be observed, on occasion, in CT − 1 as well as CT + 1 and CT + 2. Moreover, responses within an individual sometimes differed across the circa-CT trials (e.g., severe-intensity behavior being observed in CT + 1 but not CT + 2; Fig. 3C). Nevertheless, the majority of participants' responses in the CT + 1 and CT + 2 trials were indicative of heavy-intensity domain behavior. In the CT + 2 trial, at most only 3–5 participants produced demonstrably severe-intensity responses for each variable, and the identity of these participants also varied. The sensitivity of the individual analysis is supported by the contrasting results of the S1 analysis: only one response for arEMG and three for $m\dot{V}O_2$ were misclassified as heavy exercise (Figs. 3A and 3B). All of the fatigue and torque complexity responses were correctly classified as severe. Such a low occurrence of severe-intensity responses in the CT + 1 and CT + 2 trials was unexpected, and thus, these data demonstrated that the CT is not a sudden threshold. Instead, the confidence limits associated with this parameter seem to reflect a band of physiological uncertainty, wherein different system states can coexist and the responses observed cannot be predicted (i.e., a continuous phase transition (2,18).

Physiological basis of the phase transition. The data of the present study demonstrate that the transition from heavy-intensity to severe-intensity behavior seems to occur gradually over ~5% of the MVC. This is illustrated in Figure 4, which presents rate of change in the potentiated doublet torque in this study and each of our previous investigations of the CT concept. That such a transition was collectively reflected in markers of muscle activation, muscle metabolic rate, indices of neuromuscular fatigue, and torque complexity, with none of these changes preceding the others implies that the underlying mechanism(s) of this transition is likely to be common to all of these factors. Importantly, although most responses within 2 SE of the CT were likely to be characterized as heavy-intensity exercise, severe-intensity responses were observed, albeit rarely, in all trials. This implies that the

responses to exercise "at" CT/CP (i.e., within the 95% confidence limits of CT/CP) are unpredictable, which is supported by the inconsistent responses to exercise performed at CP reported previously (6,14,15). It is known that the CP is correlated with the proportion of type I muscle fibers (9), and it has been shown repeatedly that the CP is sensitive to O_2 delivery (30,31). Moreover, CP is correlated with indices of muscle capillary density, particularly around type I muscle fibers (13). Most recently, it has been shown that CP declines after prolonged exercise (32), a decline that is blunted by ingestion of carbohydrates (33). Thus, CT/CP seems to be determined, in part, by muscle fiber properties and the supply of substrates and O_2 to them, and therefore, the mechanism(s) responsible for the phase transition we propose here likely involves these factors too.

It is known that there are spatial and temporal heterogeneities in muscle metabolism (34–36) and muscle oxygenation (37), the latter reflecting the dynamic balance between microvascular O_2 delivery and mitochondrial O_2 consumption (Heinonen et al. (38) for review). Thus, those factors above thought to determine the CP are subject to spatial and temporal variation in activity and substrate supply. We suggest that such heterogeneities may also explain the apparent phase transition that occurs around CT. Specifically, the band of intensities represented by the CT is, by definition, a region wherein the neuromuscular system is precariously balanced between heavy and severe exercise. Subtle variations in motor unit recruitment, metabolism, and blood flow within trials, or between days, may result in the system being tripped into the severe domain when it would be expected to be in the heavy domain and vice versa. Indeed, James and Green (39) produced realistic power–duration curves during constant load and all-out exercise by modeling the effect of fatigue on the motor unit pool. The importance of this is that the aforementioned heterogeneities could increase the probability of motor units developing functionally significant levels of fatigue, leading to the recruitment of additional, and even less fatigue-resistant motor units. In other words, the phase transition we propose is a small region of the intensity spectrum in which the probability of unsustainable fatigue and resulting non–steady-state behavior developing significantly increases. However, it is only in the severe domain itself (outside the confidence limits of the CT/CP) that this probability reaches unity.

Limitations. The use of an isometric contraction model and NIRS-derived measures of $m\dot{V}O_2$ lead naturally to limitations in the extent to which we can generalize the current findings. Specifically, although the $m\dot{V}O_2$ measures produce qualitatively reasonable profiles of the $m\dot{V}O_2$ response, they represent the interrogation of a relatively small and superficial volume of the vastus lateralis (20,22). The same limitation is true for the measurement of arEMG, drawn again from the vastus lateralis. The use of cuff occlusion to produce these measurements further limited the present protocol inasmuch as the measurements of fatigue-related variables could only take place before and at the immediate cessation of exercise. We were not able to track the time course of fatigue, central or peripheral, although such measurements have been made previously in the heavy and severe domains (2). Finally, the lack of a condition exclusively in the heavy intensity domain, as mentioned previously, was chosen to limit the already considerable experimental burden on the participants. Thus, we had to compare other conditions to the CT − 2 condition as a proxy for heavy-intensity exercise. We believe that this choice was justified, as Figure 4 shows that the rate of peripheral fatigue development was identical to our previous studies in which contractions were performed demonstrably below the CT.

CONCLUSIONS

The present study demonstrated that the CT is not a sudden physiological threshold, based on the responses to isometric contractions within the confidence limits of the CT parameter estimate. Specifically, when exercise was performed two SE above the point estimate of CT (~107% of CT), most of the observed responses seemed to be in the heavy-intensity domain. Such a low occurrence of severe-intensity responses suggests that the CI values themselves represent a region of physiological uncertainty, which we suggest is caused either by CT varying day-to-day by an amount represented by the confidence limits or that heterogeneities in muscle recruitment, metabolism, and/or blood flow can "trip" the system into a different domain of behavior at any intensity within the confidence limits. In either case, we contend that the CT is most accurately defined as a continuous phase transition rather than a sudden threshold. Therefore, to ensure consistent physiological responses, exercise tasks should be performed outside the confidence limits associated with the CT or CP parameter estimate.

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The authors report no competing interests.

The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation, and results of the present study do not constitute endorsement by the American College of Sports Medicine.

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